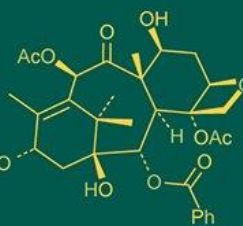
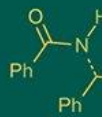


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In vitro evaluation of fungicides and biocontrol agents against *Phytophthora infestans* causing potato late blight

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Abstract

Late blight, caused by the oomycete *Phytophthora infestans*, is a major constraint to potato production, necessitating effective and sustainable disease-management strategies. The present study evaluated the antagonistic activity of seven *Trichoderma* isolates and two bacterial isolates against *P. infestans* and assessed the efficacy of eleven fungicidal treatments under *in vitro* conditions. Microbial antagonists were evaluated using volatile-compound and dual-culture assays, whereas fungicides were tested at 10 and 25 ppm using the poisoned-food technique. Considerable variation was observed among the biocontrol agents. In the volatile-compound assay, *Leucobacter* sp. P14 showed the highest inhibition of pathogen growth (57.30%), followed by *Ochrobactrum* sp. P1 (56.87%) and *Trichoderma* sp. Th7 (54.76%) after 12 days. In the dual-culture assay, *Trichoderma* sp. Th7 exhibited the greatest inhibition (57.04%), followed by Th3 (50.19%), P1 (49.81%), Th6 (49.26%) and *T. harzianum* Th2 (48.70%). Fungicides exhibited markedly higher inhibitory activity than the microbial antagonists. Oxathiapipronil + mancozeb, azoxystrobin + tebuconazole, ethaboxam and ametoctrobin + dimethomorph completely inhibited pathogen growth (100%) at both concentrations. Fluopicolide + propamocarb hydrochloride produced 95.69% and 96.12% inhibition at 10 and 25 ppm, respectively, after 12 days. Among protectant fungicides, copper hydroxide showed comparatively greater activity, whereas propineb was least effective. These findings identify promising microbial antagonists and fungicidal treatments for further validation under pot and field conditions and their potential integration into sustainable late-blight management.

Keywords: Potato, late blight, *Phytophthora infestans*, *Trichoderma*, bacterial antagonists, biocontrol, fungicides

Introduction

Potato (*Solanum tuberosum* L.) is one of the world's most important food crops and is cultivated across diverse agroecological environments, with increasing importance for food security, rural livelihoods and agro-industrial processing. Potato production has expanded substantially in developing countries because of its high productivity, relatively short crop duration and adaptability to different production systems (Birch *et al.*, 2012) [3]. However, sustainable potato production is constrained by numerous biotic stresses, among which diseases caused by oomycetes, fungi, bacteria and viruses remain major limitations to productivity and tuber quality. In particular, late blight continues to represent a major challenge because of its rapid epidemic development and capacity to cause severe losses under conducive environmental conditions (Mishra *et al.*, 2026) [21].

Late blight is caused by the oomycete *Phytophthora infestans* (Mont.) de Bary and is widely regarded as the most destructive disease of potato. The pathogen can infect leaves, stems and tubers and favourable combinations of temperature, moisture and host susceptibility can facilitate rapid epidemic development (Fry). The epidemiological success of *P. infestans* is strongly associated with its ability to produce abundant sporangia that are efficiently dispersed and capable of generating zoospores under suitable conditions (Judelson and Blanco, 2005) [16]. The disease therefore remains a major threat to potato production, with substantial economic losses arising from both yield reduction and the intensive measures

required for disease suppression (Haverkort *et al.*, 2008) [15]. The pathogenic success of *P. infestans* is further associated with its complex reproductive biology and considerable evolutionary potential. Populations containing compatible A1 and A2 mating types can undergo sexual reproduction and produce oospores, whereas asexual reproduction enables rapid multiplication and dissemination during epidemics (Grünwald and Flier, 2005) [10]. The genome of *P. infestans* is characterized by extensive repetitive regions and a large repertoire of secreted effector genes, features that contribute to its ability to adapt to host resistance and changing selection pressures (Haas *et al.*, 2009) [12]. During infection, effector proteins manipulate host cellular processes and interfere with plant immune responses, thereby facilitating pathogen colonization and disease development (Raffaele *et al.*, 2010) [25].

The management of late blight consequently remains difficult, particularly because pathogen populations can overcome host resistance and adapt to disease-control measures. The intimate interaction between *P. infestans* and potato involves specialized infection structures and effector-mediated manipulation of host cellular processes, allowing the pathogen to evade or suppress multiple layers of plant defence. Chemical fungicides therefore remain an important component of late-blight management because they can provide relatively rapid and effective suppression when applied appropriately (Cooke *et al.*, 2011) [6]. However, repeated use of fungicides with specific modes of action can impose strong selection pressure on pathogen populations and contribute to the development of reduced sensitivity or resistance.

The emergence of reduced fungicide efficacy has been documented for several compounds used against *P. infestans*, demonstrating the importance of monitoring pathogen sensitivity and selecting effective active ingredients for disease management. Reduced efficacy of fluazinam, for example, has been associated with changes in the sensitivity of *P. infestans* populations, highlighting the evolutionary challenge associated with repeated fungicide use (Schepers *et al.*, 2018) [26]. Sustainable chemical management therefore depends on the judicious selection and integration of fungicides with different modes of action rather than reliance on repeated applications of a single chemistry (Cooke *et al.*, 2011) [6]. Consequently, comparative screening of available fungicides under controlled conditions can provide valuable preliminary information for identifying compounds with strong inhibitory activity against the pathogen.

In parallel, biological control has emerged as an important complementary approach for reducing dependence on synthetic fungicides. Several bacterial and fungal antagonists have demonstrated activity against *P. infestans* through mechanisms including antibiosis, competition, production of antimicrobial metabolites, enzymatic activity and induction of host defence responses (Wang and Long, 2023) [28]. Soil-associated *Bacillus* and *Pseudomonas* spp. have shown antagonistic activity against *P. infestans*, with some strains producing metabolites capable of directly inhibiting pathogen growth and development (Caulier *et al.*, 2018) [4]. Similarly, *Pseudomonas fluorescens* LBUM636 produces phenazine-1-carboxylic acid, which can interfere with *P. infestans* growth and late-blight development (Morrison *et al.*, 2017) [22].

Fungal antagonists also possess considerable potential for biological suppression of late blight. *Trichoderma atroviride*, for example, has demonstrated inhibitory activity against *P. infestans* under controlled experimental conditions, supporting its potential as a component of integrated disease management (Al-Mughrabi, 2008) [1]. Nevertheless, the effectiveness of biological control agents is often inconsistent because their performance can depend on strain characteristics, environmental conditions, application timing and interactions with the host and pathogen (Hashemi *et al.*, 2022) [14]. Recent multi-location evaluations further demonstrate that although selected biological control agents can reduce late-blight development, their efficacy may remain lower than that of conventional fungicides, emphasizing the importance of screening and optimizing promising candidates before field deployment (Pöldmets *et al.*, 2025) [23].

Despite extensive research on individual fungicides and microbial antagonists, their relative efficacy can vary with pathogen population, chemical active ingredient, microbial strain and experimental conditions. Consequently, systematic *in vitro* screening provides an important preliminary step for comparing candidate treatments and identifying those with strong inhibitory potential against *P. infestans*. Such assays can also provide mechanistic indications of direct antagonism and pathogen growth suppression, thereby supporting subsequent greenhouse and field evaluation. Therefore, the present study was undertaken to screen selected fungicides and biocontrol agents against *Phytophthora infestans* under *in vitro* conditions, with the objective of identifying effective candidates for their potential incorporation into sustainable and integrated late-blight management strategies.

Materials and Methods

Experimental site and study period

The study was conducted during 2025-2026 at the ICAR-Central Potato Research Institute (CPRI), Regional Station, Modipuram, Meerut, Uttar Pradesh, India and Research work was planned at the Centre of Excellence for Sanitary and Phytosanitary, Department of Plant Pathology, Sardar Vallabhbhai Patel University of Agriculture and Technology (SVPUAT), Meerut. The study comprised isolation and maintenance of *Phytophthora infestans*, evaluation of microbial biocontrol agents and *in vitro* screening of fungicides against the pathogen.

Plant material and pathogen isolation

Potato (*Solanum tuberosum* L.) cultivar Kufri Bahar was used as the host material for collection of late-blight-infected samples. Potato leaves and tubers showing characteristic late-blight symptoms were collected from potato fields and transported to the laboratory under cool conditions. Symptomatic tissues were washed under running tap water to remove adhering soil and debris and dried with sterile blotting paper. Approximately 0.5-cm tissue pieces were excised from the advancing margins of lesions and surface-disinfested with 1% sodium hypochlorite (NaOCl) for 30 s. The tissues were subsequently rinsed twice with sterile distilled water for 15 s and dried aseptically. The disinfested tissue pieces were transferred onto rye agar medium and incubated at 18±2 °C. Developing colonies were examined periodically and hyphal tips showing morphological characteristics consistent with *P. infestans*

were transferred aseptically to fresh rye agar medium. Purification was achieved through repeated hyphal-tip transfer and the resulting cultures were maintained on rye agar medium for subsequent experiments.

Preparation of rye agar medium

Rye agar medium was used for isolation, purification and maintenance of *P. infestans*. Rye grains were thoroughly washed and processed to obtain a rye extract. The extract was filtered through sterile cheesecloth, followed by incorporation of agar and sucrose and the volume was adjusted with distilled water. The medium was sterilized by autoclaving at 121 °C and 15 psi for 15-20 min. After sterilization, the medium was cooled to approximately 45-50 °C and poured aseptically into sterile Petri plates.

Morphological identification of *Phytophthora infestans*

Putative *P. infestans* cultures were identified based on colony morphology and microscopic characteristics. Sporangia were examined microscopically for their characteristic hyaline, papillate and lemon-shaped to ovoid morphology. Sporangioophores were examined for sympodial branching and hyphal and sporangial characteristics were recorded. Where sporulation was observed, cultures were subjected to low-temperature treatment to facilitate zoospore release and subsequently examined microscopically. Identification was based on established morphological characteristics of *P. infestans*.

Microbial biocontrol agents

Seven *Trichoderma* isolates (Th1-Th7) and two bacterial isolates (P1 and P14) were obtained from the culture collection maintained at the Biological Control Research Laboratory, ICAR-CPRI Regional Station, Modipuram, Meerut. The *Trichoderma* isolates were maintained on Potato Dextrose Agar (PDA) slants, whereas the bacterial isolates were maintained on Nutrient Agar Medium (NAM) and King's B medium, as applicable. Cultures were periodically subcultured to maintain viability and purity before use. The microbial treatments comprised *Trichoderma* sp. Th1 (CPRI RS-M-Th1), *Trichoderma harzianum* Th2 (CPRI RS-M-Th2), *T. harzianum* Th3 (CPRI RS-M-Th3), *T. harzianum* Th4 (CPRI RS-M-Th4), *Trichoderma afroharzianum* Th5 (CPRI RS-M-Th5), *Trichoderma* sp. Th6 (CPRI RS-M-Th6), *Trichoderma* sp. Th7 (CPRI RS-M-Th7), *Ochrobactrum* sp. P1 (CPRI RS-M-P1) and *Leucobacter* sp. P14 (CPRI RS-M-P14). The untreated *P. infestans* culture served as the control.

Evaluation of microbial antagonists using volatile compounds

The effect of volatile compounds produced by the microbial antagonists on *P. infestans* was evaluated using a paired-plate assay based on the method of Dennis and Webster (1971). For *Trichoderma*, a 5-mm-diameter mycelial disc obtained from a 4-6-day-old culture was placed centrally on PDA plates and incubated at 24±1 °C for 96 h. The bacterial isolates were grown on their respective media, with P1 cultured on King's B medium and P14 on NAM plates and incubated at 30±1 °C for 48 h. A 5-mm-diameter disc of *P. infestans* was placed centrally on rye agar medium. The pathogen-containing plate was inverted over the antagonist-containing plate to allow exposure to volatile compounds without direct physical contact between the cultures. The

paired plates were sealed with Parafilm and incubated at 18±1 °C. Plates containing *P. infestans* without an antagonist served as controls. Radial growth of the pathogen was recorded after 12 days of incubation.

Evaluation of microbial antagonists using dual culture

The antagonistic activity of the microbial isolates against *P. infestans* was evaluated using a dual-culture assay. A 5-mm-diameter mycelial disc of *P. infestans*, obtained from a 12-day-old culture, was placed on one side of a 90-mm Petri plate containing approximately 20 mL of rye agar medium. A 5-mm-diameter disc of a 3-day-old *Trichoderma* culture was placed on the opposite side at an approximate distance of 6 cm between the inoculum. Because of the comparatively slower growth of *P. infestans*, the pathogen was inoculated 4-5 days before introduction of the *Trichoderma* isolate. Plates were incubated at 18±2 °C. For bacterial antagonists, a 5-mm-diameter disc of *P. infestans* was placed on the solidified medium, while the bacterial antagonist was streaked on the opposite side of the plate. P1 and P14 were evaluated on King's B medium and NAM, respectively. The bacterial antagonists were introduced 4-5 days after inoculation of the pathogen. Plates containing *P. infestans* alone were maintained as controls. Radial growth was recorded after 6 and 12 days of inoculation.

Assessment of pathogen growth inhibition

The radial growth of *P. infestans* was measured along two perpendicular axes and the mean colony diameter was calculated. Percentage inhibition of pathogen growth was calculated relative to the untreated control using the following equation

$$I(\%) = \frac{C - T}{C} \times 100$$

Where *I* represent percentage inhibition, *C* represents radial growth of *P. infestans* in the untreated control and *T* represents radial growth in the treatment.

In vitro evaluation of fungicides

The efficacy of selected fungicides against *P. infestans* was evaluated using the poisoned-food technique. Eleven fungicidal treatments were tested at 10 and 25 ppm. A 1% stock solution was prepared by dissolving 0.1 g of each formulated fungicide in 10 mL of sterile distilled water. For the 10-ppm concentration, 100 µL of the stock solution was incorporated into 100 mL of molten rye agar medium, whereas 250 µL of the stock solution was incorporated into 100 mL of medium to obtain 25 ppm. The amended medium was thoroughly mixed and poured into sterile Petri plates. Rye agar medium without fungicide served as the untreated control. Following solidification, a 5-mm-diameter mycelial disc of *P. infestans* was placed centrally on each plate. The plates were incubated at 18±1 °C and radial growth was recorded after 6 and 12 days of inoculation. Percentage inhibition of pathogen growth was calculated using the equation described above. The fungicide treatments were copper oxychloride 50% WP, chlorothalonil 78.12% WP, mancozeb 75% WP, propineb 70% WP, copper hydroxide 53.8% DF, zineb 75% WP, oxathiapipronil 0.6% + mancozeb 60% WP, azoxystrobin 11% + tebuconazole 18.3% SC, ethaboxam 40% SC, ametoctrodin 27% + dimethomorph 20.27% and fluopicolide 5.56% +

propamocarb hydrochloride. All fungicides were evaluated at 10 and 25 ppm.

Experimental design and statistical analysis

The fungicide experiment consisted of 12 treatments, including the untreated control, with three replications per treatment and was arranged in a Completely Randomized Design (CRD). The microbial antagonist assays were similarly conducted using replicated plate assays, with *P. infestans* alone serving as the control. Data on radial growth and percentage inhibition were subjected to Analysis of Variance (ANOVA). Treatment means were compared using the Critical Difference (CD) at the 5% probability level. Percentage inhibition data were subjected to appropriate transformation before statistical analysis when required, while original means are presented for interpretation.

Results

Antagonistic activity of microbial biocontrol agents against *Phytophthora infestans*

The tested microbial antagonists exhibited differential inhibitory activity against *P. infestans* in both volatile-compound and dual-culture assays. The magnitude of inhibition varied among isolates and between the two assay

systems, indicating differences in their antagonistic potential.

Inhibition of *P. infestans* by volatile compounds

Volatile compounds produced by the microbial antagonists significantly inhibited the radial growth of *P. infestans* after 12 days of incubation (Table 1; Fig. 1; Plate 1). The untreated control recorded a radial growth of 5.91 cm. Among the nine microbial treatments, P14 (T₉; CPRI RS-M-P14) exhibited the greatest inhibitory effect, restricting pathogen growth to 2.53 cm and resulting in 57.30% inhibition. P1 (T₈; CPRI RS-M-P1) was the next most effective treatment, with a radial growth of 2.55 cm and 56.87% inhibition. Th7 (T₇) and Th2 (T₂) resulted in radial growth of 2.68 and 2.85 cm, corresponding to 54.76 and 51.80% inhibition, respectively. Th1 (T₁) restricted pathogen growth to 3.20 cm, representing 45.88% inhibition. The remaining *Trichoderma* isolates also reduced pathogen growth relative to the control. Th4 (T₄), Th3 (T₃), Th6 (T₆) and Th5 (T₅) resulted in radial growth of 3.33, 3.64, 3.71 and 3.83 cm, corresponding to 43.77, 38.48, 37.21 and 35.31% inhibition, respectively. The reported critical difference at the 5% probability level was 0.24 cm. T₇ and T₈ were statistically comparable with T₉, whereas the remaining treatments differed significantly from the most effective treatment.

Table 1: Inhibition of the radial growth of the *P. infestans* *in-vitro*, by volatile compounds released by different fungal and bacterial bio agents

Treatments	Inhibition after 12 days	
	Radial growth of the Pathogen (in cm)	% Inhibition
T ₁ -CPRI RS-M-Th1	3.2	45.88
T ₂ -CPRI RS-M-Th2	2.85	51.80
T ₃ -CPRI RS-M-Th3	3.64	38.48
T ₄ -CPRI RS-M-Th4	3.33	43.77
T ₅ -CPRI RS-M-Th5	3.83	35.31
T ₆ -CPRI RS-M-Th6	3.71	37.21
T ₇ -CPRI RS-M-Th7	2.68	54.76
T ₈ -CPRI RS-M-P1	2.55	56.87
T ₉ -CPRI RS-M-P14	2.53	57.30
Control	5.91	-
SE (m)	0.08	-
CD (5%)	0.24	-

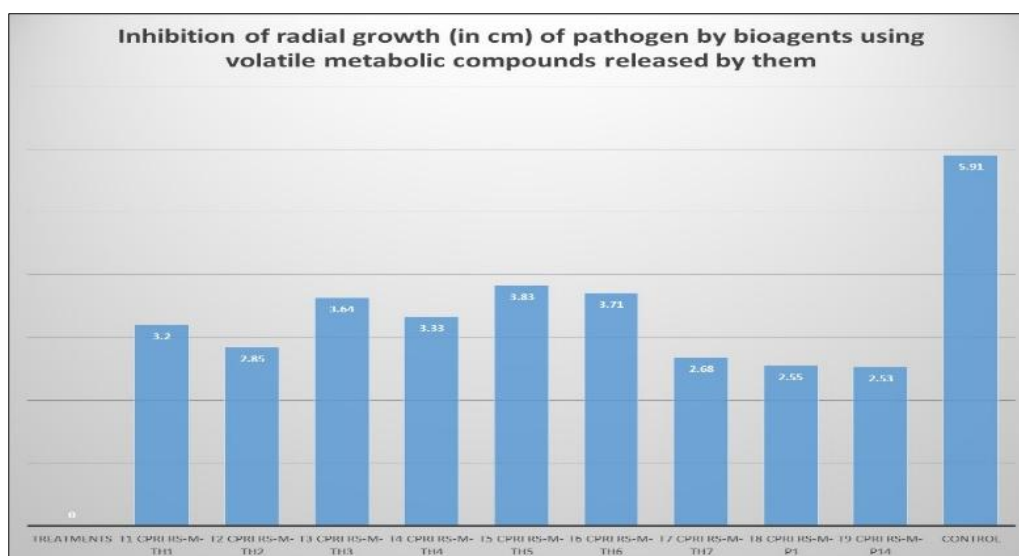


Fig 1: Inhibition of the radial growth of the *P. infestans* *in-vitro*, by volatile compounds released by different bio agents of *Trichoderma* spp. and bacterial isolates, 12 days after treatment

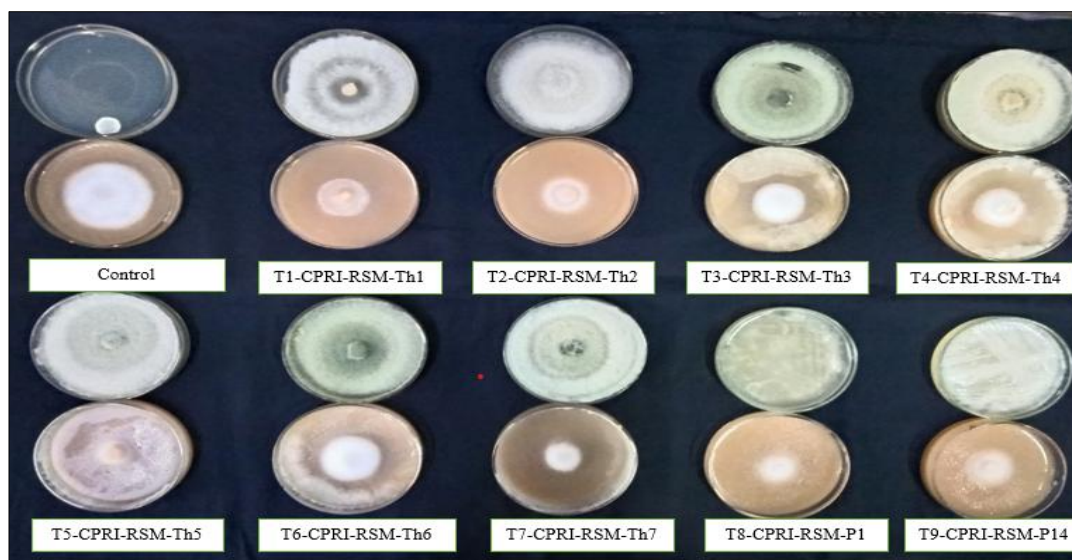


Plate 1: Inhibition of the radial growth of the *P. infestans in-vitro*, by volatile compounds released by different bio agents of *Trichoderma spp.* and bacterial isolates, 12 days after treatment

Inhibition of *P. infestans* by dual culture

Direct confrontation with the microbial antagonists resulted in significant inhibition of *P. infestans*, although the relative efficacy of individual isolates changed with incubation period (Table 2; Fig. 2; plate 2). After 6 days of incubation, the untreated control recorded a radial growth of 6.37 cm. Th2 (T₂; CPRI RS-M-Th2) was the most effective antagonist, restricting pathogen growth to 4.28 cm and producing 32.72% inhibition. This was followed by Th4 (T₄), Th1 (T₁), P1 (T₈) and Th6 (T₆), which resulted in radial growth of 4.75, 4.83, 4.87 and 4.88 cm, corresponding to 25.39, 24.08, 23.56 and 23.30% inhibition, respectively. Th3 (T₃) and Th5 (T₅) each resulted in 5.00 cm radial growth and 21.47% inhibition. P14 (T₉) produced 5.12 cm radial growth and 19.63% inhibition, whereas Th7 (T₇) was the least inhibitory at this observation time, with 5.68 cm radial growth and 10.73% inhibition. The CD at the 5%

probability level was 0.52 cm. After 12 days, the inhibitory response increased for most antagonists. The control recorded a radial growth of 9.00 cm, whereas Th7 (T₇) exhibited the greatest inhibition, restricting pathogen growth to 3.87 cm and resulting in 57.04% inhibition. Th3 (T₃), P1 (T₈), Th6 (T₆) and Th2 (T₂) followed, with radial growth values of 4.48, 4.52, 4.57 and 4.62 cm and corresponding inhibition values of 50.19, 49.81, 49.26 and 48.70%, respectively. Th5 (T₅), Th1 (T₁) and Th4 (T₄) resulted in radial growth of 5.28, 5.40 and 5.58 cm, corresponding to 41.30, 40.00 and 37.96% inhibition, respectively. P14 (T₉) showed comparatively lower inhibition, with a radial growth of 6.10 cm and 32.78% inhibition. The CD at the 5% probability level was 0.23 cm. Thus, Th7 showed the highest antagonistic activity after 12 days, whereas Th2 was the most effective isolate at the earlier observation period.

Table 2: Inhibition of the radial growth of the *P. infestans in-vitro*, by dual culture technique by different bio agents of fungal and bacteria

Treatments	Inhibition after 6 days		Inhibition after 12 days	
	Radial growth of the Pathogen (in cm)	% Inhibition	Radial growth of the Pathogen (in cm)	% Inhibition
T ₁ -CPRI RS-M-Th1	4.83	24.08	5.40	40.00
T ₂ -CPRI RS-M-Th2	4.28	32.72	4.62	48.70
T ₃ -CPRI RS-M-Th3	5.00	21.47	4.48	50.19
T ₄ -CPRI RS-M-Th4	4.75	25.39	5.58	37.96
T ₅ -CPRI RS-M-Th5	5.00	21.47	5.28	41.30
T ₆ -CPRI RS-M-Th6	4.88	23.30	4.57	49.26
T ₇ -CPRI RS-M-Th7	5.68	10.73	3.87	57.04
T ₈ -CPRI RS-M-P1	4.87	23.56	4.52	49.81
T ₉ -CPRI RS-M-P14	5.12	19.63	6.10	32.78
Control	6.37	-	9.00	-
SE (m)	0.17	-	0.08	-
CD (5%)	0.52	-	0.23	-

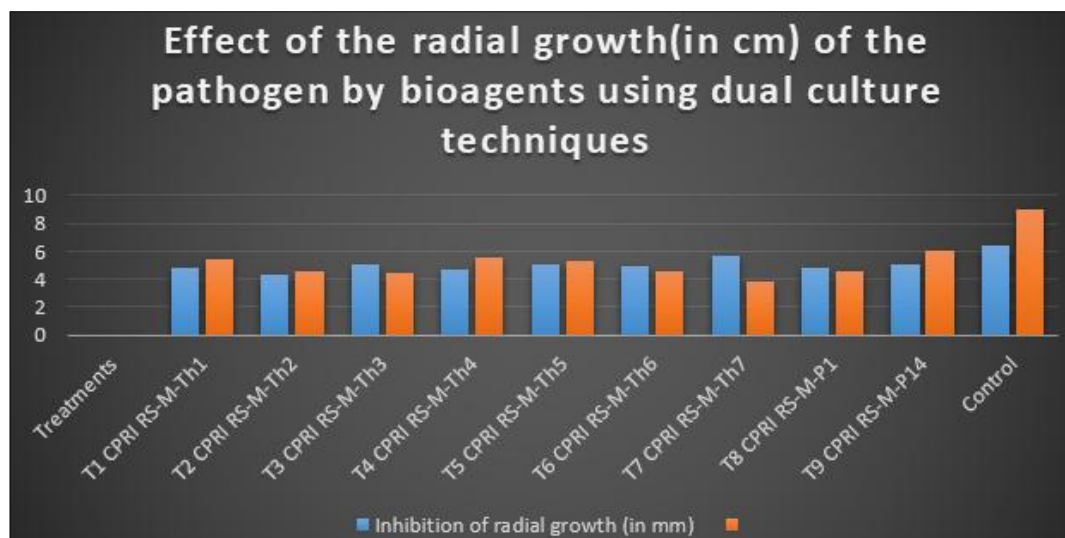


Fig 2: Inhibition of the radial growth of the *P. infestans* *in-vitro*, by dual culture technique by different bio agents of *Trichoderma* spp. and bacterial isolates

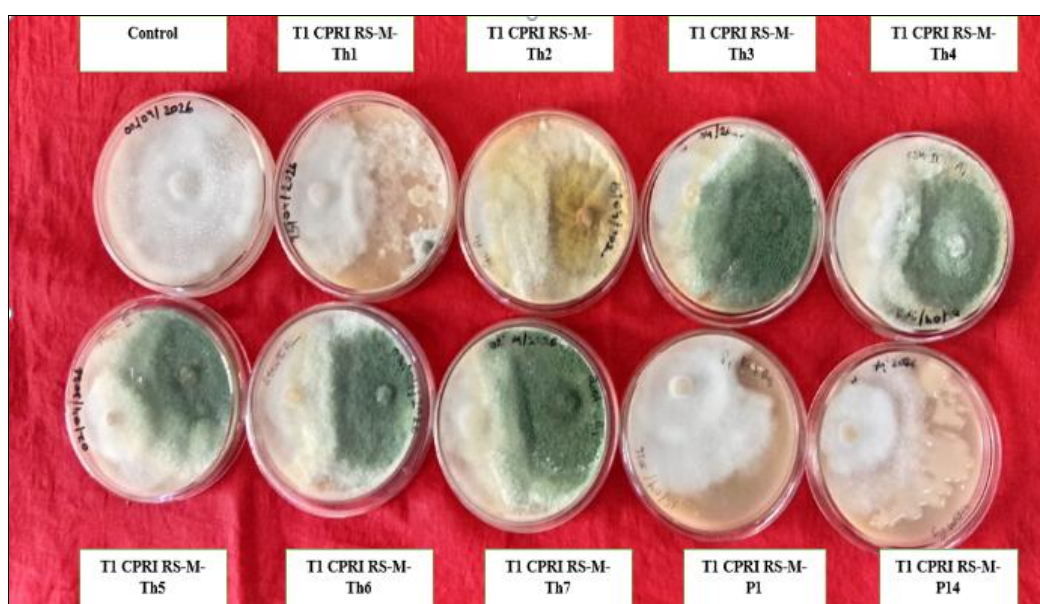


Plate 2: Inhibition of the radial growth of the *P. infestans* *in-vitro*, by dual culture technique by different bio agents of *Trichoderma* spp. and bacterial isolates.

Efficacy of fungicides against *P. infestans* at 10 ppm

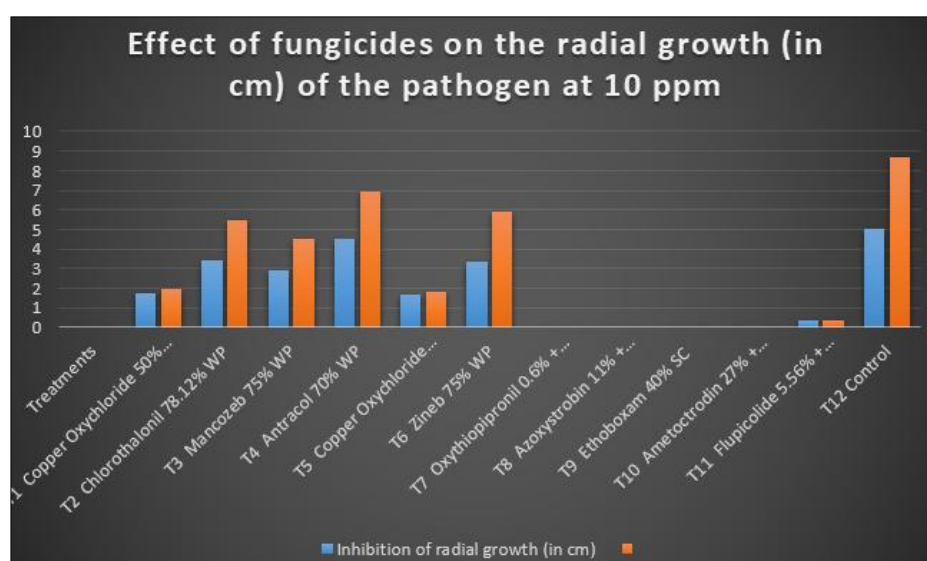
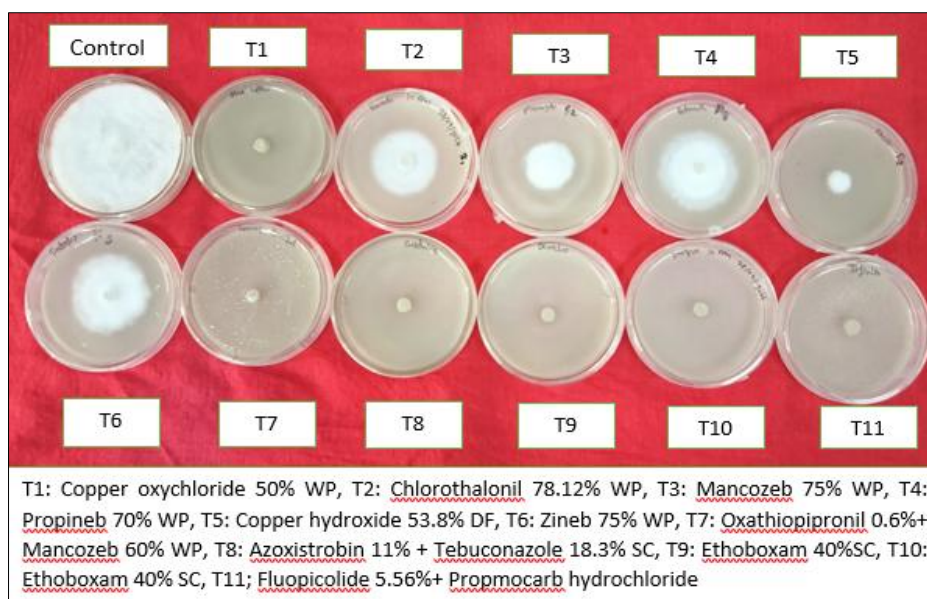
The fungicides differed substantially in their inhibitory activity against *P. infestans* at 10 ppm (Table 3; Fig. 3; Plate 3). Four treatments, namely oxathiopiprolin + mancozeb (T₇), azoxystrobin + tebuconazole (T₈), ethaboxam (T₉) and ametoctrodin + dimethomorph (T₁₀), completely suppressed radial growth of the pathogen at both 6 and 12 days, corresponding to 100% inhibition. After 6 days, the untreated control recorded 5.01 cm radial growth. Fluopicolide + propamocarb hydrochloride (T₁₁) strongly inhibited pathogen growth, with radial growth of 0.38 cm and 92.52% inhibition. Copper hydroxide (T₅) and copper oxychloride (T₁) resulted in radial growth of 1.66 and 1.75 cm, corresponding to 66.84 and 65.09% inhibition, respectively. Mancozeb (T₃), zineb (T₆) and chlorothalonil

(T₂) produced 41.90, 33.67 and 32.18% inhibition, respectively. Propineb (T₄) showed the lowest activity, with 4.54 cm radial growth and 9.49% inhibition.

After 12 days, the untreated control reached 8.71 cm radial growth. T₇, T₈, T₉ and T₁₀ continued to provide complete inhibition of pathogen growth. T₁₁ maintained high efficacy, restricting radial growth to 0.38 cm and producing 95.69% inhibition. Copper hydroxide (T₅) and copper oxychloride (T₁) resulted in radial growth of 1.83 and 1.95 cm, corresponding to 78.95 and 77.67% inhibition, respectively. Mancozeb (T₃), chlorothalonil (T₂), zineb (T₆) and propineb (T₄) showed comparatively lower inhibition of 47.90, 37.43, 32.55 and 20.35%, respectively. The CD values were 0.13 and 0.18 cm at 6 and 12 days, respectively, indicating significant differences among fungicide treatments.

Table 3: Percent inhibition of the radial growth of *P. infestans* by *in-vitro* poison food technique at 10 ppm at different days interval

Treatments	Inhibition after 6 days		Inhibition after 12 days	
	Radial growth of the Pathogen (in cm)	% Inhibition	Radial growth of the Pathogen (in cm)	% Inhibition
T ₁ -Copper Oxychloride 50% WP	1.75	65.09	1.95	77.67
T ₂ -Chlorothalonil 78.12% WP	3.40	32.18	5.45	37.43
T ₃ -Mancozeb 75% WP	2.91	41.90	4.54	47.90
T ₄ -Propineb 70% WP	4.54	9.49	6.94	20.35
T ₅ -Copper hydroxide 53.8% DF	1.66	66.84	1.83	78.95
T ₆ -Zineb 75% WP	3.33	33.67	5.88	32.55
T ₇ -Oxythiopyrrol 0.6% + Mancozeb 60% WP	0.00	100.00	0.00	100.00
T ₈ -Azoxystrobin 11% + Tebuconazole 18.3% SC	0.00	100.00	0.00	100.00
T ₉ -Ethoboxam 40% SC	0.00	100.00	0.00	100.00
T ₁₀ -Ametoctrocin 27% + Dimethomorph 20.27%	0.00	100.00	0.00	100.00
T ₁₁ -Flupicolide 5.56% + Propamocarb Hydrochloride	0.38	92.52	0.38	95.69
T ₁₂ Control	5.01	-	8.71	-
SE (m)	0.04	-	0.07	-
CD (5%)	0.13	-	0.18	-

**Fig 3:** Inhibition of the radial growth of the *P. infestans*, by *in-vitro* poison food technique using fungicides at 10 ppm concentration**Plate 3:** Inhibition of the radial growth of the *P. infestans*, by *in-vitro* poison food technique using fungicides at 10 ppm concentration

Efficacy of fungicides against *P. infestans* at 25 ppm

Increasing the fungicide concentration to 25 ppm generally increased pathogen suppression compared with the corresponding 10 ppm treatments (Table 4; Fig. 4; Plate 4). T₇, T₈, T₉ and T₁₀ completely inhibited radial growth of *P. infestans* at both observation periods, recording 100% inhibition. After 6 days, radial growth in the untreated control was 6.08 cm. T₁₁ restricted pathogen growth to 0.20 cm, corresponding to 96.71% inhibition. Among the remaining fungicides, copper hydroxide (T₅) was the most effective, with 1.32 cm radial growth and 78.36% inhibition, followed by copper oxychloride (T₁), chlorothalonil (T₂), zineb (T₆) and mancozeb (T₃), which produced radial growth of 1.48, 1.60, 1.90 and 2.53 cm and corresponding inhibition of 75.62, 73.70, 68.77 and 58.36%, respectively.

Propineb (T₄) showed the lowest activity among the fungicides, with 4.17 cm radial growth and 31.51% inhibition. After 12 days, T₇-T₁₀ continued to completely suppress pathogen growth. T₁₁ maintained strong inhibitory activity, with radial growth of 0.30 cm and 96.12% inhibition. Copper hydroxide (T₅) resulted in the highest inhibition among the remaining single-active fungicides, restricting radial growth to 1.53 cm and producing 80.17% inhibition. Copper oxychloride (T₁) and chlorothalonil (T₂) resulted in 78.66 and 78.02% inhibition, respectively, whereas zineb (T₆) produced 66.16% inhibition. Mancozeb (T₃) showed 48.92% inhibition with 3.95 cm radial growth, while propineb (T₄) remained the least effective treatment, with 5.92 cm radial growth and 23.49% inhibition. The CD values were 0.21 and 0.20 cm at 6 and 12 days, respectively.

Table 4: Percentage Inhibition of the radial growth of *P. infestans* by *in-vitro* poison food technique using fungicides at 25 ppm at different days' interval

Treatments	Radial growth & Inhibition & after 6 days		Radial growth & Inhibition after 12 days	
	Radial growth of the Pathogen (in cm)	% Inhibition	Radial growth of the Pathogen (in cm)	% Inhibition
T ₁ -Copper Oxychloride 50% WP	1.48	75.62	1.65	78.66
T ₂ -Chlorothalonil 78.12% WP	1.60	73.70	1.7	78.02
T ₃ -Mancozeb 75% WP	2.53	58.36	3.95	48.92
T ₄ -Propineb 70% WP	4.17	31.51	5.92	23.49
T ₅ -Copper hydroxide 53.8% DF	1.32	78.36	1.53	80.17
T ₆ -Zineb 75% WP	1.9	68.787	2.62	66.16
T ₇ -Oxythiopyronil 0.6% + Mancozeb 60% WP	0.00	100.00	0.00	100.00
T ₈ -Azoxystrobin 11% + Tebuconazole 18.3% SC	0.00	100.00	0.00	100.00
T ₉ -Ethoboxam 40% SC	0.00	100.00	0.00	100.00
T ₁₀ -Ametocrodin 27% + Dimethomorph 20.27%	0.00	100.00	0.00	100.00
T ₁₁ -Flupicolide 5.56% + Propamocarb Hydrochloride	0.20	96.71	0.3	96.12
T ₁₂ Control	6.08	-	7.73	-
SE (m)	0.07	-	0.07	-
CD (5%)	0.21	-	0.2	-

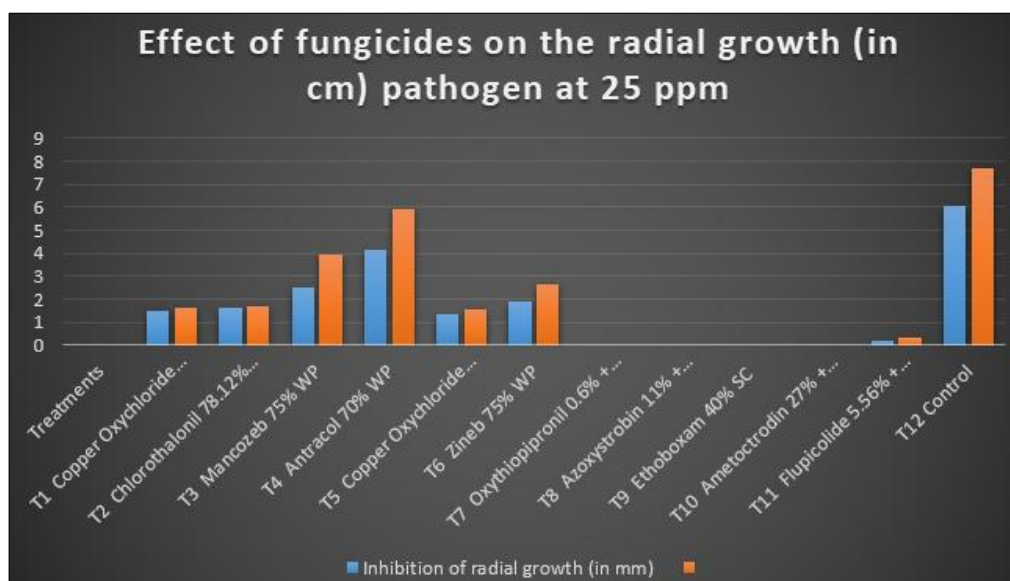


Fig 4: Inhibition of the radial growth of *P. infestans* by *in-vitro* poison food technique using fungicides at 25 ppm concentration

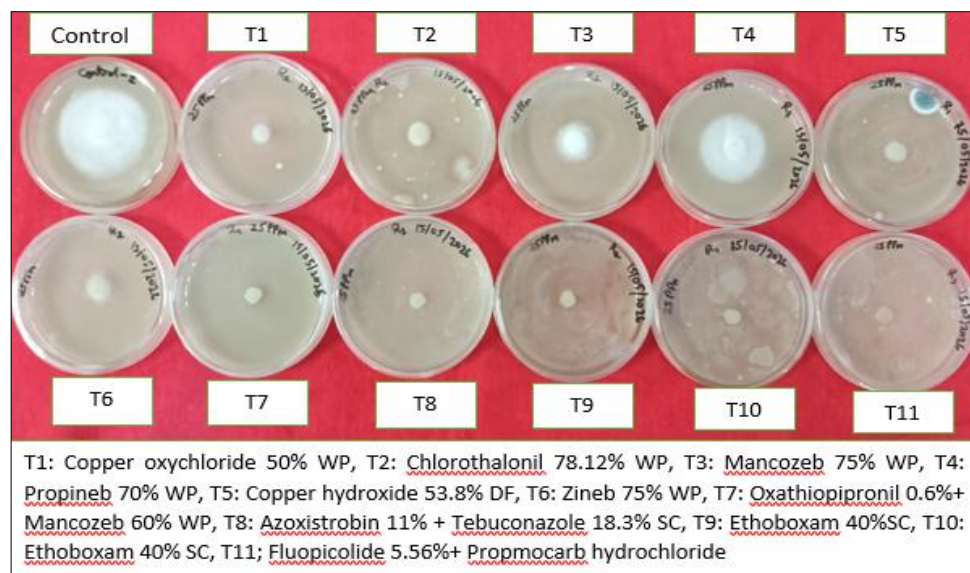


Plate 4: Inhibition of the radial growth of *P. infestans* by *in-vitro* poison food technique using fungicides at 25 ppm concentration

Comparative efficacy of microbial antagonists and fungicides

The efficacy of the microbial antagonists varied according to the antagonistic mechanism evaluated. In the volatile-compound assay, P14 (T₉) produced the highest inhibition after 12 days (57.30%), followed by P1 (T₈; 56.87%) and Th7 (T₇; 54.76%). In the dual-culture assay, Th7 produced the highest inhibition after 12 days (57.04%), whereas Th2 was the most effective antagonist at 6 days (32.72%). The fungicides exhibited considerably greater inhibitory activity than the microbial antagonists under the tested *in vitro* conditions. Oxathiopiprolin + mancozeb, azoxystrobin + tebuconazole, ethaboxam and ametocrodin + dimethomorph completely suppressed pathogen growth at both 10 and 25 ppm. Fluopicolide + propamocarb hydrochloride also exhibited strong activity, producing more than 92% inhibition at both concentrations. Among the single-active fungicides, copper hydroxide and copper oxychloride consistently showed comparatively higher inhibitory activity than mancozeb, zineb, chlorothalonil and propineb.

Discussion

The present study demonstrated considerable variation among the microbial antagonists in their ability to suppress *Phytophthora infestans* under *in vitro* conditions. In the volatile-compound assay, all seven *Trichoderma* isolates and both bacterial isolates inhibited pathogen growth relative to the untreated control, although the magnitude of inhibition varied among isolates. *Leucobacter* sp. P14 produced the highest inhibition (57.30%), closely followed by *Ochrobactrum* sp. P1 (56.87%), whereas *Trichoderma* sp. Th5 showed the lowest inhibition (35.31%). Such isolate-dependent variation is characteristic of microbial biocontrol systems because antagonistic activity depends on the metabolic capacity, growth behaviour and secondary-metabolite profile of individual strains (Harman *et al.* 2004) [13]. The greater inhibition produced by P14 and P1 in the volatile assay suggests that airborne or diffusible volatile metabolites may have contributed to suppression of *P. infestans*. Microbial VOCs can inhibit phytopathogens without requiring direct physical contact and represent an important mechanism of microbial antagonism (Vinale *et al.* 2008) [27]. However, because the present study did not

chemically characterize the volatile compounds, the results demonstrate volatile-mediated inhibition but do not establish the identity or specific biological activity of individual metabolites. This distinction is important because VOC production can vary considerably with microbial strain, culture medium, temperature and physiological stage (Kanchiswamy *et al.* 2015) [17]. The performance of *Trichoderma* isolates in the volatile assay also differed considerably. Th7 and Th2 were among the most effective isolates, producing 54.76 and 51.80% inhibition, respectively, whereas Th5 produced 35.31% inhibition. Similar strain-dependent differences in antagonistic activity have been reported for *Trichoderma*, indicating that species-level identification alone may not adequately predict biocontrol performance (Guzmán-Guzmán *et al.* 2019) [11]. Recent research on *Trichoderma*-*P. infestans* interactions has likewise demonstrated substantial variation in pathogen suppression among *Trichoderma* strains (Cournault *et al.* 2025) [7]. Thus, the superior performance of particular isolates in the present study provides a rational basis for selecting strain-specific candidates for further evaluation. The dual-culture assay demonstrated a different pattern of antagonistic activity. At 6 days after inoculation, Th2 produced the highest inhibition of *P. infestans* (32.72%), whereas at 12 days Th7 became the most effective isolate, producing 57.04% inhibition. Th3, P1 and Th6 also showed comparatively high inhibition at 12 days. The increase in inhibition with increasing incubation period for several isolates indicates that direct antagonism may become progressively stronger as the antagonist establishes itself in the culture environment. *Trichoderma* spp. are known to suppress phytopathogens through competition for nutrients and space, antibiosis and mycoparasitism (Benítez *et al.* 2004) [2]. Mycoparasitism represents one of the major mechanisms through which *Trichoderma* can directly suppress pathogenic fungi and oomycetes. During this interaction, *Trichoderma* hyphae recognize and interact with pathogen structures and can produce hydrolytic enzymes such as chitinases, β -glucanases and proteases (Zeilinger and Omann 2007) [29]. The relatively high inhibition produced by Th7 at 12 days may therefore be associated with more effective direct interaction or metabolite production. Nevertheless, because the present experiment

did not measure hydrolytic enzyme activity or microscopically confirm hyphal coiling and penetration, these mechanisms should be regarded as possible explanations rather than experimentally demonstrated mechanisms.

The bacterial isolates also exhibited antagonistic activity, although their relative performance differed between the two assays. P14 was the strongest antagonist in the volatile assay but was comparatively less effective in the dual-culture assay at 12 days. In contrast, P1 maintained comparatively high inhibition in both assays. This difference indicates that the antagonistic potential of bacterial isolates may depend on whether inhibition occurs through volatile metabolites or direct interaction. Plant-associated bacteria can suppress pathogens through antibiosis, siderophore production, competition and other secondary metabolites (Raaijmakers and Mazzola 2012) [24]. Therefore, the contrasting responses of P1 and P14 between assay systems may reflect differences in their predominant antagonistic mechanisms. The fungicide assays produced substantially greater inhibition of *P. infestans* than the microbial antagonists. At 10 ppm, oxathiapipronil + mancozeb, azoxystrobin + tebuconazole, ethaboxam and ametoctrodin + dimethomorph completely inhibited radial growth at both 6 and 12 days. Fluopicolide + propamocarb hydrochloride also showed very high activity, producing 92.52 and 95.69% inhibition at 6 and 12 days, respectively. The persistence of complete inhibition by four treatments indicates high sensitivity of the tested *P. infestans* isolate to these fungicide chemistries under the experimental conditions. The strong activity of oxathiapipronil-containing treatment is consistent with the highly specific activity of oxathiapipronil against oomycete pathogens. Oxathiapipronil targets oxysterol-binding protein and exhibits strong activity against *Phytophthora* species (Cohen *et al.* 2018) [5]. Ethaboxam has also demonstrated strong inhibitory activity against *P. infestans* and other oomycete pathogens (Kim *et al.* 2004) [19]. The complete inhibition observed for these treatments in the present study is therefore consistent with their recognized biological activity against oomycetes. Increasing the fungicide concentration from 10 to 25 ppm generally increased the efficacy of the conventional fungicides. Copper hydroxide increased from 66.84 to 78.36% inhibition at 6 days, while copper oxychloride increased from 65.09 to 75.62%. At 12 days, copper hydroxide produced 80.17% inhibition and copper oxychloride produced 78.66% inhibition. In contrast, propineb remained the least effective treatment, producing only 23.49% inhibition at 25 ppm after 12 days. Variation in fungicide efficacy against *P. infestans* has also been reported among different active ingredients and concentrations (Khadka *et al.* 2020) [18]. The high efficacy of modern oomycete-active fungicides should not, however, be interpreted as evidence that systemic or target-specific fungicides are universally superior to protectant fungicides. Fungicide performance depends on the active ingredient, target site, concentration and sensitivity of the pathogen population. Moreover, *in vitro* inhibition does not necessarily translate directly into equivalent field efficacy because plant penetration, environmental conditions, fungicide persistence and epidemic development are not represented in culture-plate assays (Forbes 2012) [8]. The strong activity of several target-specific fungicides also has implications for resistance management. Oxathiapipronil

resistance has been associated with changes affecting its target site in *P. infestans*, demonstrating the potential for resistance development under selection pressure (Kong *et al.* 2021) [20]. Consequently, highly effective fungicides should be incorporated into resistance-management programmes involving appropriate rotation or mixtures with fungicides having different modes of action (FARC 2025). Overall, the present study identified complementary microbial and chemical candidates for further evaluation against late blight. P14 and P1 were the most effective microbial antagonists in the volatile assay, whereas Th7 showed the highest inhibition in the 12-day dual-culture assay. Among the fungicides, oxathiapipronil + mancozeb, azoxystrobin + tebuconazole, ethaboxam and ametoctrodin + dimethomorph completely inhibited pathogen growth at both tested concentrations. These findings provide a strong *in vitro* basis for selecting promising treatments for subsequent greenhouse and field validation.

Conclusion

The present study demonstrated substantial variation among the tested microbial biocontrol agents and fungicides in suppressing *Phytophthora infestans* under *in vitro* conditions. All seven *Trichoderma* isolates and both bacterial isolates exhibited inhibitory activity against the pathogen, although their efficacy varied considerably according to the antagonistic mechanism evaluated. In the volatile-compound assay, *Leucobacter* sp. P14 (T₉; CPRI RS-M-P14) showed the highest inhibition of pathogen growth (57.30%), followed by *Ochrobactrum* sp. P1 (56.87%) and *Trichoderma* sp. Th7 (54.76%). In contrast, the dual-culture assay identified *Trichoderma* sp. Th7 (T₇; CPRI RS-M-Th7) as the most effective antagonist, producing 57.04% inhibition after 12 days, followed by Th3, P1, Th6 and *T. harzianum* Th2. These contrasting responses between the two assays indicate that the inhibitory potential of the tested microorganisms may depend on the mode of antagonism and the interaction established with the pathogen. The fungicide assays revealed pronounced differences in the sensitivity of *P. infestans* to the tested active ingredients and formulations. Oxathiapipronil + mancozeb, azoxystrobin + tebuconazole, ethaboxam and ametoctrodin + dimethomorph completely inhibited pathogen growth at both 10 and 25 ppm. Fluopicolide + propamocarb hydrochloride also exhibited very high inhibitory activity, with 95.69% and 96.12% inhibition at 10 and 25 ppm, respectively, after 12 days. Among the protectant fungicides, copper hydroxide and copper oxychloride showed comparatively greater inhibitory activity than propineb, while mancozeb, chlorothalonil and zineb exhibited intermediate efficacy. The consistently lower efficacy of propineb, particularly at the lower concentration, suggests comparatively reduced activity against the tested *P. infestans* isolate under the conditions of the assay. Overall, the findings establish that both microbial antagonists and fungicides can substantially suppress *P. infestans* growth *in vitro*, but their efficacy is dependent on the antagonist, fungicide chemistry, concentration and assay conditions. The strong performance of selected *Trichoderma* and bacterial isolates provides a basis for further evaluation of their biocontrol potential, while the highly active fungicide treatments could serve as candidate components for integrated late-blight management. However, *in vitro* inhibition alone does not establish field efficacy; therefore,

the most promising microbial and chemical treatments should be validated under controlled pot and field conditions before their incorporation into an integrated disease-management programme.

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Declarations of Competing Interests

The authors declare that they have no competing interests that could have influenced the research described in this paper.

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